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Original article

Impact of obstructive sleep apnea in recruitment of coronary collaterality during inaugural acute myocardial infarction

Impact du syndrome d'apnée du sommeil sur le développement de la circulation coronaire collatérale en cas d'infarctus du myocarde inaugural

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Abstract

Background. – Obstructive sleep apnea (OSA) may lead to myocardial preconditioning by increasing coronary collateral vessel recruitment in patients with acute coronary occlusion.

Aim. – To determine the relationship between the severity of obstructive sleep apnea and coronary collaterality during acute myocardial infarction.

Methods. – This study prospectively included 71 patients with an inaugural myocardial infarction who had undergone a coronary angiography within 24 h of onset. All patients underwent an overnight polygraph before discharge and were classified according to the apnea–hypopnea index (AHI). Coronary collaterals were scored by visual analyses and according to the Rentrop grading system.

Results. – Mean age was 59 ± 11 years and 83% of patients were men. All patients had complete or subtotal occlusion of the infarct-related artery. After the sleep study, patients were divided into two groups: 25 were suffering from OSA (AHI > 15/h). Patients with OSA showed better collateral vessel development (Rentrop score ≥ 1) compared to non-OSA patients (68 vs. 41%, $P = 0.032$). AHI was significantly higher in patients with developed coronary collaterals (Rentrop ≥ 1) compared to those without collaterality (17.74 ± 13.2 vs. 12.24 ± 10.9, $P = 0.025$).

Conclusion. – Coronary collateral development may be increased in OSA patients who are presenting with a first myocardial infarction.

Keywords: Obstructive sleep apnea; Coronary circulation; Myocardial infarction; Apnea–hypopnea index

Résumé

Introduction. – Le syndrome d'apnées obstructives du sommeil (SAOS) peut contribuer à un pré-conditionnement du myocarde en augmentant le développement de la circulation coronaire collatérale chez les patients présentant une occlusion coronaire aiguë.

Objectif. – Déterminer la relation entre la sévérité du SAOS et la collatéralité coronaire en cas d'infarctus aigu du myocarde.

Méthodes. – Cette étude prospective a inclus 71 patients ayant présenté un infarctus du myocarde inaugural et ayant bénéficié d'une coronarographie dans les 24 heures. Tous les patients ont eu un enregistrement polygraphique et ont été classés en fonction de l'indexe apnée–hypopnée (IAH). Le développement de la circulation coronaire collatérale a été analysé selon la classification de Rentrop.

Abbreviation: AHI, Apnea hypopnée index; IRA, Infarct-related artery; OSA, Obstructive sleep apnea; PCI, Primary coronary intervention; STEMI, ST elevation myocardial infarction; TIMI, Thrombolysis in myocardial infarction.

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Résultats. – L'âge moyen de la population était de 59 ± 11 ans avec 83 % de sexe masculin. Tous les patients avaient une occlusion complète ou subtotale de l'artère coronaire responsable de l'infarctus. Un SAOS (IAH > 15/h) a été identifié chez 25 patients. Une collatéralité coronaire (Rentrop ≥ 1) était plus fréquente chez les patients apnéiques par rapport aux autres patients (68 vs 41 %, $p = 0{,}032$). L'IAH était significativement plus élevée chez les patients ayant une circulation coronaire collatérale (Rentrop ≥ 1) par rapport à ceux qui n'ont pas de collatéralité (17,74 ± 13,2 vs 10,9 ± 12,24, $p = 0{,}025$).

Conclusion. – Le développement de la circulation coronaire collatérale peut être augmenté chez les patients atteints de syndrome d'apnées obstructives du sommeil se présentant avec un infarctus du myocarde inaugural.

Mots clés : Apnée obstructive du sommeil ; Circulation coronaire ; Infarctus du myocarde ; Index apnée–hypopnée

1. Background

Obstructive sleep apnea (OSA) is characterized by repetitive airflow reduction or temporary cessation of breathing caused by upper airway collapse, resulting in intermittent oxygen desaturation. There is increasing evidence that OSA is a risk factor for the development of cardiovascular disease, including myocardial infarction and stroke [1,2]. Furthermore, severe OSA has a negative prognostic impact on early and late outcomes after myocardial infarction [3].

The underlying pathophysiological mechanisms involve a complex interplay between intermittent nocturnal hypoxemia, sympathetic activation, endothelial dysfunction, and the release of pro-inflammatory cytokines [4–6].

In contrast to the deleterious effects of OSA, a protective role has also been described. A study has suggested that nocturnal cycles of repeated hypoxemia–reoxygenation may lead to myocardial ischemic preconditioning, conferring protection from acute coronary events [7]. In a recent observational study [8], infarct size, measured by peak troponin levels, was significantly lower in patients with OSA compared to those without sleep apnea. Steiner was the first to support this hypothesis, by showing that patients with OSA had better coronary collateral vessel development [9].

Coronary collaterals are anastomotic connections without an intervening capillary bed between the segments of the same coronary artery or different coronary arteries. In cases of acute myocardial infarction, the presence of coronary collaterals may help preserve the viability of myocardium until mechanical or pharmacological coronary reperfusion.

To our knowledge, no studies have been reported on the impact of OSA and the development of coronary collaterals in patients with inaugural acute myocardial infarction. In our study, we hypothesize that OSA is associated with better recruitment of coronary collaterals in patients with a first acute myocardial infarction and a total occlusion of an infarct-related coronary artery (IRA).

2. Methods

2.1. Study design and population

This prospective study included patients who presented with a ST segment elevation myocardial infarction (STEMI) between April 2011 and March 2012. STEMI was defined as an ischemic symptom that lasted for > 30 min, elevated cardiac troponin T levels, and a ST segment elevation of ≥ 1 mm in at least two contiguous limb leads, or ≥ 2 mm in the precordial leads.

Patients admitted to our institution with a first STEMI and who had undergone a coronarograph for primary percutaneous coronary intervention (PCI) or to control fibrinolysis within 24 h of onset were included in the study. Non-inclusion criteria included a previous known diagnosis of OSA, electrical instability, cardiogenic shock, previous coronary events, previous documented myocardial ischemia, or an inability to give informed consent. Spontaneous or pharmacological recanalization of the infarct-related artery, defined as TIMI (thrombolysis in myocardial infarction) of grade 3 flow as assessed in the initial coronary angiogram was an angiographic exclusion criteria [10]. Patients with a central or mixed apnea syndrome, discovered after the sleep study, were also excluded. All patients provided their written informed consent.

2.2. Overnight sleep study

All patients underwent an overnight polygraph during their hospital stay, after leaving the coronary care unit and before discharge (not exceeding 15 days post-STEMI). The sleep studies were done in the Department of Cardiology with optimized conditions of sleep: in a quiet room without taking sleeping pills or sedatives.

The polygraph recordings were performed between 9:00 pm and 6:00 am using a portable diagnostic device (Medibyte Junior 2.0, Braebon, Ontario, Canada). The parameters measured included nasal airflow, thoraco-abdominal movements, arterial oxygen saturation (pulse oximetry), and heart rate. Outputs from the portable diagnostic device were analyzed by an investigator blinded to the clinical characteristics of the patient.

Respiratory events were defined according to recommendations of the American Academy of Sleep Medicine [11]. An apneic episode was defined as cessation of airflow for > 10 s and hypopnea as a > 50% reduction in airflow lasting > 10 s. An event was also considered to be hypopnea when a reduction in airflow did not reach the 50% criteria but was associated with > 3% desaturation of arterial oxygen.

Obstructive apnea was defined as the absence of air flow despite respiratory movement. Central sleep apnea was defined as the absence of both air flow and respiratory movement.

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The apnea–hypopnea index (AHI) was calculated as the number of apneas and hypopneas per hour of recording time in bed. An AHI of 15 events per hour was considered clinically significant. Daytime sleepiness was evaluated using the Epworth Sleepiness Scale [12].

2.3. Coronary angiographyy

Coronary angiograms were evaluated by two interventional cardiologists blinded to the clinical data and to the results of the sleep study. All angiographic films were acquired at 15 frames per second.

The TIMI anterograde flow of the infarct-related artery was assessed [10]. The score of the collateral circulation was based on the view that best illustrated the occluded vessel. The presence of coronary collaterals was defined and visually assessed according to the Cohen and Rentrop classification[13]:

- grade 0 = no filling of collateral vessels;
- grade 1 = filling of collateral vessels without any opacification of the epicardial recipient artery;
- grade 2 = partial filling of the target epicardial artery by collateral vessels;
- grade 3 = complete epicardial filling of the recipient artery by collaterals.

A coronary collateral presence was considered as the presence of grade 1, 2, or 3.

All patients received a bolus injection of unfractionated heparin (0.5 mg/kg) following oral administration of aspirin (250 mg) and clopidogrel (600 mg). Primary PCI was performed according to conventional methods.

2.4. Statistical analyses

The results are reported as means ± SD for continuous variables or as percentages for categorical data. The analyses of categorical variables were performed using Fisher's exact test. For continuous data, the unpaired Student's *t*-test was used to compare groups. The Wilcoxon rank sum test was used to assess the relationship between AHI and the presence of coronary collateral vessels.

All statistical analyses were carried out using the statistical Package for Social Sciences (SPSS 18.0 for Windows). A *P* value of ≤ 0.05 was considered statistically significant.

3. Results

During the study period, 196 patients presented with STEMI; of these, 119 were eligible and 43 met the angiographic exclusion criteria (TIMI-3 flow in the infarct-related artery). Three patients refused the sleep study and two patients presented with central sleep apnea as assessed by a polygraph. Thus, a total of 48 patients were excluded from the study, with the final study group consisting of the remaining 71 patients.

Eighty-three percent of the patients were men. The mean age was 59 ± 11 years. Hypertension was present in 30 patients, 32 patients had a history of diabetes, and 53 were current smokers. Thirteen patients had received prior fibrinolytic therapy and were referred to our center for coronarography, and 58 were admitted for primary PCI. The mean symptom-to-angiography time was 8 ± 6 h. The baseline demographic and clinical characteristics of this study population are shown in Table 1.

All patients had complete or subtotal occlusion (TIMI < 3) of the IRA: this was represented by the left anterior descending artery in 52%, the left circumflex artery in 14%, and the

Table 1
Patients' demographic and clinical characteristics.

	Overall ($n = 71$)	Non-OSA (AHI ≤ 15) $n = 46$	OSA (AHI > 15) $n = 25$	*P* value
Age (years)	59 ± 11	57 ± 11	62 ± 11	0.048
Male gender	59 (83%)	38 (82.6%)	21 (84%)	0.88
Hypertension	30 (42%)	18 (39%)	12 (48%)	0.47
Smoker	53 (74%)	35 (76%)	18 (72%)	0.7
Diabetes	32 (45%)	20 (43%)	12 (48%)	0.71
Dyslipidemia	17 (24%)	11 (24%)	6 (24%)	0.71
Weight (kg)	75 ± 14	73 ± 14	79 ± 13	0.08
Height (m)	1.68 ± 12	1.67 ± 0.13	1.69 ± 0.10	0.48
BMI (kg/m^2)	26.9 ± 5.8	26.51 ± 6.4	27.81 ± 4.55	0.37
Abdominal circumference (cm)	94 ± 15	92 ± 14	99 ± 16	0.036
Neck circumference (cm)	37 ± 4	37 ± 4	37 ± 4	0.424
Systolic BP (mmHg)	125 ± 19	124 ± 17	126 ± 22	0.60
Diastolic BP (mmHg)	74 ± 13	73 ± 13	74 ± 14	0.87
Location of infarction				
Anterior STEMI	30 (42%)	20 (44%)	10 (40%)	0.77
Non-anterior STEMI	41 (58%)	26 (56%)	15 (60%)	
Fibrinolytic therapy	13 (18%)	8 (17%)	5 (20%)	0.23
Primary PCI	58 (82%)	38 (83%)	20 (80%)	0.16

BP: blood pressure; BMI: body-mass index; LVEF: left ventricular ejection fraction; PCI: primary coronary intervention; STEMI: ST segment elevation myocardial infarction.

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Table 2
Patients' angiographic and polygraphic data.

	Non-OSA (AHI ≤ 15) $n = 46$	OSA (AHI > 15) $n = 25$	P value
Symptom-to-angiography time (h)	7 ± 6	10 ± 6	0.08
IRA: LAD/CX/RCA	25/6/15	12/4/9	0.86
Rentrop 0	27 (59%)	8 (32%)	0.032
Rentrop ≥ 1	19 (41%)	17 (68%)	
3-vessel disease	8 (17%)	6 (24%)	0.78
LVEF (%)	51 ± 8	50 ± 10	0.64
Mean sat. O_2 (%)	92.97 ± 1.36	93.69 ± 1.94	0.072
Minimum sat. O_2 (%)	85.27 ± 4.8	85.94 ± 5.26	0.59
Mean AHI (events/h)	7.5 ± 3.54	28.88 ± 10.71	< 0.001
Epworth score	2 ± 1	3 ± 2	0.003

IRA: infarct-related coronary artery; LAD: left anterior descending artery; CX: circumflex artery; RCA: right coronary artery; OSA: obstructive sleep apnea; AHI: apnea–hypopnea index; LVEF: left ventricular ejection fraction.

right coronary artery in 34%. The patients' mean left ventricular ejection fraction was 50% ± 9.

No visible collateral filling was found in 35 patients. Rentrop of grade 1 was found in 14, grade 2 in 12, and grade 3 in 10 patients.

After the sleep study, patients were divided into two groups: the 25 patients who had an AHI > 15/h were classified as suffering from OSA. They were compared with the remaining 46 patients whose AHI ≤ 15/h. Angiographic and polygraphic data from the two groups are presented in Table 2. The level of daytime sleepiness, evaluated by the Epworth scale, was higher in OSA patients (3 ± 2 vs. 2 ± 1, $P = 0.003$).

There were no significant differences between the two groups in terms of age, gender, coronary risk factors, infarct location, and time to coronarography. Angiographic findings, including IRA distribution and multi-vessel diseases, were also similar between the two groups. Nevertheless, abdominal circumference was significantly higher in OSA patients than those without OSA.

There were no significant differences in mean and minimum oxygen saturation (SaO_2) during the sleep study between the two groups. However, patients with OSA showed better collateral development (Rentrop score ≥ 1) compared with non-OSA (68% vs. 41%, $P = 0.032$) patients.

In addition, patients with developed coronary collaterals (Rentrop grade ≥ 1) showed significantly higher AHI compared to those without coronary collaterals (Rentrop grade 0) (17.74 ± 13.2 vs. 12.24 ± 10.9, $P = 0.025$). Moreover, there was a parallel increase between AHI level and Rentrop grade, except for patients with a Rentrop grade of 3 (Fig. 1).

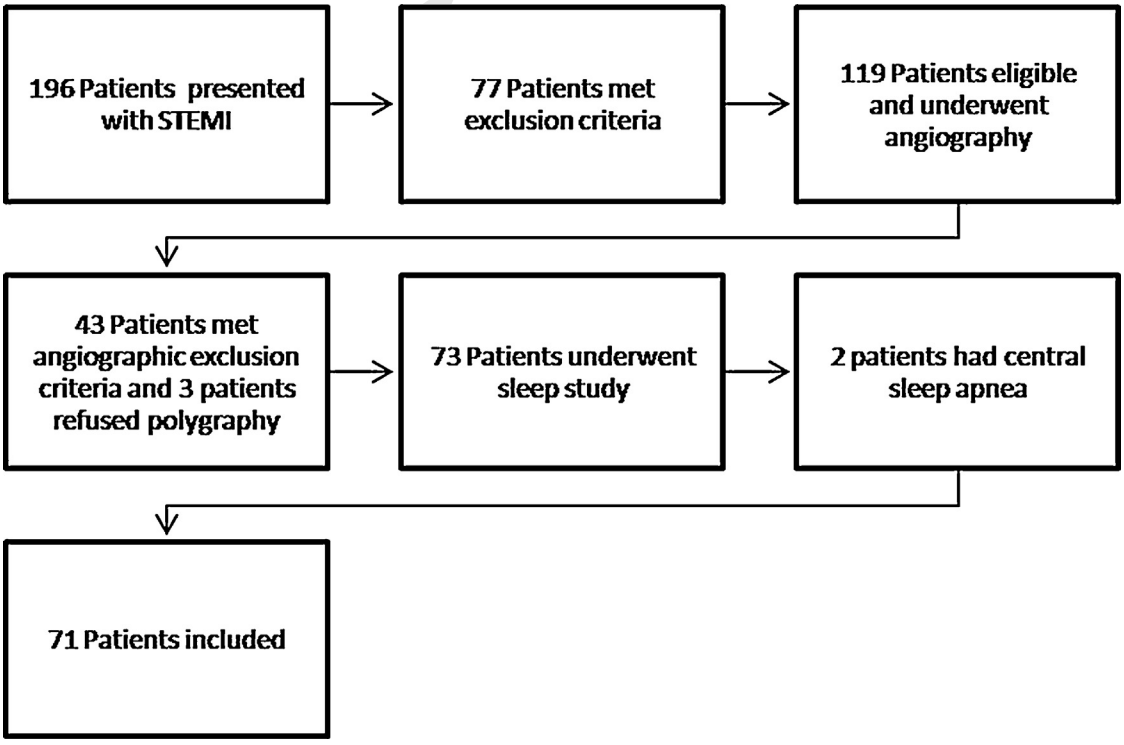

Fig. 1. Selection of the study population.

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4. Discussion

The main finding of our study is that coronary collateral development is increased in OSA patients presenting with inaugural STEMI compared with those without sleep apnea. We also found a high prevalence of previously undiagnosed OSA in patients admitted with acute myocardial infarction. These findings agree with several other studies, which also report a high prevalence of sleep apnea in patients with acute coronary syndrome [13–15]. This reinforces the idea that this problem remains under diagnosed after acute myocardial infarction [16] (Fig. 2).

Coronary arteriolar connections may already be present at birth, but may also develop during life [17]. It is well established that developed coronary collaterals may play a crucial role in the early phase of acute myocardial infarction, by limiting infarct size and preserving myocardial viability [18,19].

The most important determinant of coronary collaterals is recurrent myocardial ischemia. In fact, the burden of cardiac ischemia before an acute coronary event has been strongly associated with the presence of coronary collaterals [20]. For this reason, we excluded all patients who suffered from ischemic heart disease or previous angina. Only those with inaugural myocardial infarction were included in order to eliminate any factors that could interfere with the phenomenon of ischemia preconditioning.

Steiner et al. suggested a relationship between OSA and the development of the coronary collateral circulation [9]. However, their study included a heterogeneous population with several clinical presentations and chronic total coronary occlusion (stable angina, atypical chest pain, or dyspnea), whereas our study focused on a homogeneous group admitted for a first acute myocardial infarction. It should be noted though that 45% of our patients were diabetics, so it is possible that these patients had silent ischemia or atypical symptoms of angina for few weeks or months before the onset of infarction. Therefore, they may have undergone preconditioning ischemia, and may have skewed the main selection criteria for this study. Because there was no difference according to the prevalence of diabetes in both groups with or without sleep apnea, this did not affect the comparisons between the groups.

Furthermore, in this study, the mean abdominal circumference was higher in the OSA group but who also had better collateral circulation. Abdominal obesity is one of the main components of metabolic syndrome and several studies showed that collateral vessel development is compromised in patients with a metabolic syndrome [21,22]. Nevertheless, in our study, despite the negative impact of metabolic disorders on the development of collateral vessels, abdominal obesity did not seem to affect the coronary collaterality development, suggesting that the influence of OSA on the recruitment of collateral circulation prevails.

In addition, the chronic intermittent hypoxia caused by OSA in obese patients has been reported as being one of the underlying mechanisms in the morbidity-mortality paradox of obesity [23]. However, our findings suggest a parallel increase between the development of the collateral circulation and the severity of OSA, as quantified by AHI, except for the grade 3 Rentrop group. This latter finding was probably due to the low number of patients within this group.

In this study, all patients underwent an angiography within the first 24 h, but at very different times from the onset of pain. This disparity in delay may be responsible for the heterogeneity found in the dynamic process of coronary collateral recruitment. In fact, angiographic studies that have evaluated fibrinolytic therapy in STEMI have shown that collateral coronary vessels that are initially absent become apparent within two weeks following permanent coronary occlusion [24].

The mechanisms and factors associated with the development of coronary collaterals in this context are not totally understood. Different circulating chemokines triggered by chronic intermittent hypoxia in OSA patients have been involved in angiogenesis. The most studied factor is vascular endothelial growth factor, which can stimulate coronary collateralization in response to hypoxia [25–27]. Furthermore, patients presenting with STEMI and sleep-disordered breathing showed a higher mobilization of endothelial progenitor cells and increased expression of vascular endothelial growth factor compared to patients with normal breathing [28].

Unfortunately, in our study, which included patients in the early phase of STEMI, no measurements of angiogenic factors were performed, which could have confirmed the biological link between nocturnal cycles of hypoxia–reoxygenation and collateral development.

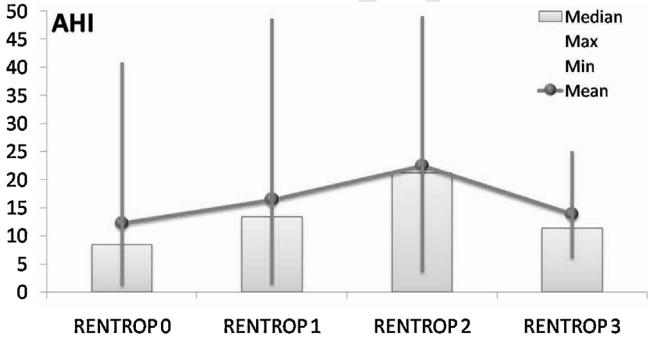

Fig. 2. AHI values according to the Rentrop grade.

5. Study limitations

There are several limitations in this study. Firstly, the number of patients was too small to allow definitive analysis of this complex phenomenon. In addition, other parameters, such as the cumulative time of desaturation or the total duration of hypoxemia, heart rate acceleration, and the arousal index, were not determined; hence, our interest in a polysomnographic study versus a polygraphic study. In fact, the depth and duration of hypoxemia and the frequency of the "ups" and "downs" of oxygen arterial saturation may play a role in the mechanisms of ischemic preconditioning.

In addition, performing sleep studies a few days after STEMI may reduce some false-positive diagnoses of OSA due to detecting transient sleep disorders during an acute coronary

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event. However, this limitation does not change our comparison between the two groups [13]. The angiographic grading of coronary collaterals using the Rentrop score can only identify vessels that are larger than 100 μm in diameter; however, the majority of collaterals are smaller. Also, angiography does not assess the distribution of collateralization within the myocardium. Nevertheless, these limitations affect the scores of all patients in the same way and, therefore, have no impact on our comparisons between the two groups. To have a significant difference when comparing the OSA and non-OSA group, the classical dichotomy Rentrop 0, 1 for absence of collaterality and Rentrop 2, 3 for presence of callaterality was not adopted in this study. We considered presence of collaterality if Rentrop grade was ≥ 1.

Finally, although the patients included in this study had no known history of coronary artery disease, the level of physical activity of each patient (which may contribute to the development of collaterality) was not analyzed, which is another limiting factor of this study.

6. Conclusion

These findings suggest that well-developed collaterals were more frequent in patients with OSA who are presenting with an inaugural acute myocardial infarction. Nocturnal repeated hypoxemia–reoxygenation cycles may lead to ischemic preconditioning, and may stimulate collateral vessels development. However, more studies are needed to improve our understanding of the underlying biological links. Further research concerning the behavior of coronary circulation and intermittent hypoxia in OSA patients with acute coronary occlusion is also needed.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

Uncited reference

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